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     (FILE 'HOME' ENTERED AT 14:27:04 ON 25 SEP 2004)

     FILE 'MEDLINE, BIOTECHDS, EMBASE, BIOSIS, SCISEARCH, CANCERLIT, CAPLUS'
     ENTERED AT 14:27:15 ON 25 SEP 2004
L1           236 S DAIRKEE S?/AU OR HAYWARD Z?/AU
L2         40284 S (LOSS (5A) HETEROZYGOSITY) OR LOH
L3             6 S L2 AND CHROMOSOMAL (5A) 3P24.3
L4            44 S L2 AND 3P24.3
L5            24 S L4 AND (BREAST CANCER OR BREAST TUMOR OR (BREAST (5A) TUMOR)
L6             0 S GANLEY ?/AU AND RABBITTS ?/AU
L7            29 S L1 AND L2
L8            29 S L7 AND BREAST
L9            25 S L8 AND TUMOR
L10           28 S TUMOR REOCCUR?
L11            1 S L10 AND L2
L12            5 DUP REM L5 (19 DUPLICATES REMOVED)
L13           12 DUP REM L7 (17 DUPLICATES REMOVED)
L14           10 DUP REM L9 (15 DUPLICATES REMOVED)
L15           24 S L4 AND BREAST
L16            5 DUP REM L15 (19 DUPLICATES REMOVED)
L17            9 DUP REM L4 (35 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 14:40:45 ON 25 SEP 2004

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Detailed deletion mapping of chromosome arm 3p in breast cancers: a 2-cM region on 3p14.3-21.1 and a 5-cM region on **3p24.3**-25.1 commonly deleted in tumors.

AUTHOR: Matsumoto S; Kasumi F; Sakamoto G; Onda M; Nakamura Y; Emi M

CORPORATE SOURCE: Department of Molecular Biology, Nippon Medical School, Kawasaki, Japan.

SOURCE: Genes, chromosomes & cancer, (1997 Nov) 20 (3) 268-74.
Journal code: 9007329. ISSN: 1045-2257.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199801

ENTRY DATE: Entered STN: 19980122
Last Updated on STN: 19980122
Entered Medline: 19980108

AB **Loss** of **heterozygosity** (**LOH**) on 3p is frequent in human renal cell carcinomas, lung cancers, and breast cancers. To define the region(s) on 3p that harbor presumptive **tumor** suppressor gene(s) for **breast cancer**, we examined 196 primary breast tumors for their patterns of **LOH** at 22 microsatellite marker loci distributed along this chromosome arm. Allelic loss at one or more loci was observed in 101 (52%) of these tumors. Detailed deletion mapping identified two distinct commonly deleted regions; one was localized to a 2-cM interval flanked by D3S1547 and D3S1295 at 3p14.3-21.1, and the other to a 5-cM interval flanked by D3S1286 and D3S1585 at **3p24.3**-25.1. The FHIT gene lies in the vicinity of the proximal commonly deleted region. Attempts to correlate **LOH** on 3p to clinicopathological parameters detected an association with the absence of the progesterone receptor (P = 0.0096). The results suggest that inactivation of unidentified tumor suppressor genes on 3p plays a role in the mechanism whereby hormone dependency is lost in the course of breast carcinogenesis.

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2002131023 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11861372
TITLE: Increased risk of local recurrence is associated with allelic loss in normal lobules of **breast cancer** patients.
AUTHOR: Li Zheng; Moore Dan H; Meng Zhen Hang; Ljung Britt-Marie; Gray Joe W; Dairkee Shanaz H
CORPORATE SOURCE: Geraldine Brush Cancer Research Institute, California Pacific Medical Center, San Francisco, California 94115, USA.
CONTRACT NUMBER: P50 CA-58207 (NCI)
SOURCE: Cancer research, (2002 Feb 15) 62 (4) 1000-3.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020228
Last Updated on STN: 20020403
Entered Medline: 20020327

AB Allelic losses characteristic of tumor cells, when displayed by morphologically normal terminal ductal lobular units (TDLUs) adjacent to carcinoma [G. Deng et al., Science (Wash. DC), 274: 2057-2059, 1996], may indicate an extended field of increased cancer susceptibility within the affected breast tissue. We investigated this possibility by asking whether the presence of **loss** of **heterozygosity** (**LOH**) at chromosome 3p11-26 in histologically normal TDLUs (3pLOHn) could lead to an increased risk of local tumor recurrence. We assessed LOHs in normal TDLUs adjacent to 48 informative cases of early-stage invasive **breast cancer** samples and found 3pLOHn in approximately 25% (13 of 48) of patients whose tumors had 3pLOH in this region. Our analyses suggest that the most frequent region of **LOH** is localized at **3p24.3**. We also demonstrate, using a Cox proportional hazards regression model, that the presence of 3pLOHn was the only variable significantly related to local tumor recurrence, leading to a 3.9-5.2-fold increase in the hazard ratio ($P < 0.05$). The time to recurrence was longer in such cases than in those without 3pLOHn, suggesting de novo tumor development. These data provide a strong rationale to assess histologically normal breast tissue at the margins of surgically excised cancers for molecular predictors of local recurrence after breast-conserving treatment.

hromosome 3p and **breast cancer**.

AUTHOR: Yang Qifeng; Yoshimura Goro; Mori Ichiro; Sakurai Takeo; Kakudo Kennichi
CORPORATE SOURCE: Second Department of Pathology, Wakayama Medical University School of Medicine, 811-1 Kimiidera, Wakayama 641-8509, Japan.. yang-qf@mail.wakayama-med.ac.jp
SOURCE: Journal of human genetics, (2002) 47 (9) 453-9. Ref: 81
Journal code: 9808008. ISSN: 1434-5161.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20020831
Last Updated on STN: 20021217
Entered Medline: 20021206

AB Solid tumors in humans are now believed to develop through a multistep process that activates oncogenes and inactivates tumor suppressor genes. **Loss** of **heterozygosity** at chromosomes 3p25, 3p22-24, 3p21.3, 3p21.2-21.3, 3p14.2, 3p14.3, and 3p12 has been reported in breast cancers. Retinoid acid receptor beta2 (3p24), thyroid hormone receptor beta1 (**3p24.3**), Ras association domain family 1A (3p21.3), and the fragile histidine triad gene (3p14.2) have been considered as **tumor** suppressor genes (TSGs) for **breast** cancers. Epigenetic change may play an important role for the inactivation of these TSGs. Screens for promoter hypermethylation may be able to identify other TSGs in chromosome 3p. Alternatively, use of an "epigenetic modifier" may enhance the response to another type of agent for **breast cancer**.

Other Reference Publication (9):
Matsumoto et al., "Detailed Deletion Mapping of Chromosome Arm 3p in Breast Cancers: A2-cM Region on 3p14.3-21.1 and 5-cM Region on 3p24.2-25.1 Commonly Deleted in Tumors," (1997) Genes Chromsomes Cancer vol. 20:268-274.

Previous Doc Next Doc Go to Doc#

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Saturday, September 25, 2004

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L1	dairkee-S$.in. or Li-Z$.in.	3295
☐	L2	Loss of heterozytosity or LOH	7846
☐	L3	L2 and 3p24	15
☐	L4	L3 and breast cancer	9
☐	L5	L3 and tumor re-occurance	0
☐	L6	tumor reoccurence	0
☐	L7	l3 and tumor	14
☐	L8	L4 and tumor	8
☐	L9	l2 and thyroid hormone receptor	18
☐	L10	L9 and (breast cancer or breast tumor)	12
☐	L11	(post near surgical treatment) and l10	1
☐	L12	(post near surgical treatment) and l8	1
☐	L13	L1 and l2	2

END OF SEARCH HISTORY

Science (1996) 274:2057; WO98/17828) have described LOH at 3p24 in normal and hyperplastic benign tissue adjacent to breast

L4: Entry 8 of 9 File: USPT May 29, 2001

DOCUMENT-IDENTIFIER: US 6239266 B1
TITLE: ZAP-3 tumor associated genes and their uses

Brief Summary Text (12):
Loss of heterozygosity (LOH) on 3p is frequent in human renal cell carcinomas, lung cancers, and breast cancers. A detailed map of a 5 cM region on 3p24.3-25.1 flanked by D3S1286 and D3S1585, which is commonly deleted in tumors, may be found in Matsumoto et al. (1997) Genes Chromosomes Cancer 20:268-274. A loss of heterozygosity has also been reported in human prostatic adenocarcinoma on the 3p24-26 and 3p22-12 regions of the short arm of chromosome 3 by Dahiya et al. (1997) Int J Cancer 71(1):20-25. Deletion in this region have also been reported for human cutaneous squamous cell neoplasms (Sikkink et al. (1997) J Invest Dermatol 109(6):801-805); in squamous cell carcinoma of the head and neck (Buchhagen et al. (1996) Head Neck 18(6):529-537); and in transitional cell carcinoma of the urinary bladder (Li et al. (1996) Am J Pathol 149(1):229-235).

Brief Summary Text (22):
Comparative sequence alignments indicate that ZAP-3 is a novel N-acetylgalactosaminyltransferase. The association of ZAP-3 loss of heterozygosity (LOH) with tumors and its predicted biological activity indicate that the involvement with tumorigenesis may be at the level of substrate glycosylation, possibly of cell adhesion or cell recognition molecules.

Brief Summary Text (23):
ZAP-3 forms an mRNA of approximately 4300 nt in length. It is expressed at low to moderate levels in heart, brain, placenta, lung, spleen, testes, liver, fetal brain, kidney, and skeletal muscle tissues. The chromosomal location of the human gene has been localized to 3P24. The ZAP-3 nucleic acid sequence is provided as SEQ ID NO:1, where the coding sequence extends from nt. 367 to 2284, and the encoded polypeptide sequence as SEQ ID NO:2.

Detailed Description Text (7):
LOH analysis. Primer sequences flanking polymorphic microsatellite loci were obtained from the Whitehead Genome Center Database (http://www-genome.wi.mit.edu/cgi-bin/contig/phys_map). PCR amplification, PCR product analysis, and calculation of the allelic ratios of heterozygous loci was performed essentially as described (Larson et al. (1997) Cancer Research 57:4082-4090). Tumor specimens and cell lines. Tumor specimens and matched non-involved tissue were provided by Memorial Sloan-Kettering Cancer Center. Cell Lines were provided by Memorial Sloan-Kettering Cancer Center and University of Tokyo. DNAs were extracted as previously described (Hampton et al. (1994) PNAS 91:6953-6957).

Detailed Description Text (12):
The ZAP-3 gene spans 55 kb of the human genome on chromosome 3p24. The gene and protein sequences are provided as SEQ ID NO:1. There are ten exons, as follows:

Other Reference Publication (5):
Dahiya et al., "Chromosomes 3p24-26 and 3p22-12 Loss in Human Prostatic Adenocarcinoma" (1997) Int J Cancer vol. 71(1):20-25.